Letters

Occupational Exposure to Environmental Tobacco Smoke

To the Editor.—The article by Dr Hammond and colleagues[1] appears to contain a serious error in data calculation. The authors state that passive monitors for nicotine were exposed for "1 week." Assuming this means 7 days, the monitors were exposed for a period of 168 hours. However, it appears that the authors used only 45 hours in the calculation of airborne nicotine concentration (with the possible exception of samples acquired at "fire stations"). If so, correcting this miscalculation significantly alters the conclusions with respect to potential worker risk as stated in their article and in the accompanying American Medical Association news release.

All official analysis methods of which I am aware (National Institute for Occupational Safety and Health, Occupational Safety and Health Administration, Environmental Protection Agency, American Society for Testing and Materials, International Standards Organization, Association of Official Analytical Chemists, etc) require that airborne concentration be calculated using the total air volume sampled. The justification by Hammond et al for their unorthodox data treatment (using only 45 of the 168 hours) is that "smoking was assumed to be taking place for 9 hours a day on each workday." However, it has been known for nearly 10 years that nicotine concentrations do not decay to zero quickly in the absence of smoking, whereas other measured components of environmental tobacco smoke (ETS) do.[2] For example, it has been demonstrated that nicotine desorbing from interior surfaces and cigarette butts can result in significant airborne levels in the absence of smoking in a well-ventilated, grounded aircraft.[3] In that study, average nicotine concentrations were found to be 6.0 $\mu g/m^3$ for an overnight, 8-hour sample. Further, it has been shown that nicotine adsorbs onto the outside of the passive monitor housings used by Hammond et al and continues to be sampled in the absence of other sources of nicotine.[4]

Therefore, even if no smoking occurred "after hours" in the workplaces studied by Hammond et al and even if the workplaces were unoccupied after "normal" business hours (ie, no overtime, additional workshifts, or cleaning crews), it is incontrovertible that nicotine would continue to be collected by the passive monitors. Accordingly, the fill sampling time must be used in calculating airborne nicotine concentrations. It appears that the concentrations reported by Hammond et al must be divided by 3.7 (168/45) to yield the actual concentrations in the workplaces studied. When calculated correctly, the average concentrations that result for the selected workplaces studied by Hammond et al become more consistent with the personal exposures of workers in more typical workplaces found in other studies in the 1990s.[5]

The pertinent issue in occupational exposure assessment is the actual amount of exposure (as determined by personal monitoring) that the worker receives at work, not the inappropriately adjusted weeklong concentration obtained from stationary air sampling during a time in which the worker is mostly absent. This issue can be resolved by relevant experimental investigations and objective data evaluations. Unfortunately, the article by Hammond et al does not appear to meet either of these criteria.

Michael W. Ogden, PhD
R. J. Reynolds Tobacco Co
Winston-Salem, NC

1. Hammond SK, Sorensen G, Youngstrom R, Ockene JK. Occupational exposure to environmental tobacco smoke. *JAMA*. 1995;274:956-960.
2. Nelson PR, Heavner DL, Collie BB, Maiolo KC, Ogden MW. Effect of ventilation and sampling time on environmental tobacco smoke component ratios. *Environ Sci Technol*. 1992;26:1909-1915.
3. Nelson PR, Heavner DL, Oldaker GB III. Problems with the use of nicotine as a predictive environmental tobacco smoke marker. In: *Measurement of Toxic and Related Air Pollutants: Environmental Protection Agency/Air and Waste Management Association International Symposium*. Pittsburgh, Pa: Air and Waste Management Association; 1990:550-555.
4. Ogden MW, Maiolo KC. Comparative evaluation of diffusive and active sampling systems for determining airborne nicotine and 3-ethenylpyridine. *Environ Sci Technol*. 1992;26:1226-1234.
5. Ogden MW, Davis RA, Maiolo KC, et al. Multiple measures of personal ETS exposure in a population-based survey of nonsmoking women in Columbus, Ohio. In: *Indoor Air '93—Proceedings of the 6th International Conference on Indoor Air Quality and Climate*. Helsinki, Finland: Indoor Air '93; 1993;1:523-528.

To the Editor.—The article by Dr Hammond and colleagues,[1] reporting high measured levels of nicotine in occupational settings without smoking restrictions, even exceeding those from some household studies, underscores concerns about the health risks associated with workplace exposure to ETS. To the degree that nicotine serves as a marker of ETS, these data are consistent with the finding that point estimates for the relative risks associated with workplace sources of exposure were somewhat higher than those associated with adult exposures from a spouse or other household members in our study of ETS and lung cancer among women who were lifetime never-smokers.[2]

The evidence offered by Hammond et al regarding the potentially high historical levels of exposure to important constituents of ETS among nonsmoking workers in various settings without smoking restrictions prompted us to examine more closely the effect of workplace ETS exposure on risk of lung cancer in our multicenter case-control study.[2] We selected only respondents who had ever reported working outside the home for 6 months or longer (90% of study subjects) and added terms to the multivariate logistic regression model to adjust not only for all of the covariates included in our original report (age, race, study area, education, dietary factors, family history of lung cancer, and employment in high-risk occupations), but also for other adult sources of ETS exposure (reported exposures from all household members and from social settings). The estimated odds ratios associated with reported workplace ETS exposure are presented in the Table. Selecting workers only and adjusting for other adult ETS exposure sources resulted in modestly enhancing

Requirements for Letters

Letters will be published at the discretion of the editors as space permits and are subject to editing and abridgment. Letters will be considered if they are typewritten double-spaced and do not exceed 500 words of text and five references. Please include a word count. Letters discussing a recent *JAMA* article should be received within 4 weeks of the article's publication. Letters must not duplicate other material published or submitted for publication. A signed statement for authorship criteria and responsibility, financial disclosure, copyright transfer, and acknowledgment is essential for publication. Letters not meeting these specifications are generally not considered. Letters will not be returned unless specifically requested. Also see *JAMA* Instructions for Authors (January 3, 1996). Letters may be submitted by surface mail: Letters Editor, *JAMA*, 515 N State St, Chicago, IL 60610; fax: (312) 464-5824; or E-mail: JAMA-letters@web.ama-assn.org.

Edited by Margaret A. Winker, MD, Senior Editor, and Phil B. Fontanarosa, MD, Senior Editor.

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Association Between Risk of Lung Cancer and Workplace Exposure to Cigarette Smoke Among Nonsmoking Women Who Have Ever Worked

Exposure Group	Cases (n=528)	Controls (n=1148)	Adjusted OR (95% CI)*
Ever exposed, No.	385	756	1.56 (1.21-2.02)
Duration of exposure, y			
0 (not exposed)	143	392	1.0
1-15	213	450	1.46 (1.10-1.94)
16-30	118	223	1.56 (1.13-2.16)
>30	54	83	2.08 (1.35-3.20) *P* for trend <.001

*Adjusted for age; race; study area; education; fruits, vegetables, and supplemental vitamin index; dietary cholesterol; family history of lung cancer; employment in high-risk occupations; and other adult sources of environmental tobacco smoke (any adult household exposure vs none; 2 or more hours a week of social exposure vs less). OR indicates odds ratio for women reporting any workplace exposure; CI, confidence interval.

the estimates of risk reported in our original article. The overall odds ratio associated with any reported workplace exposure increased from 1.39 in the earlier analysis to 1.56 in this analysis (Table), and the odds ratio for adenocarcinomas alone increased from 1.46 to 1.63 (95% confidence interval, 1.23 to 2.16). Each of the point estimates for number of years of workplace ETS exposure was likewise elevated from the previous analysis, with more than a twofold risk associated with 30 or more years of such exposure. The point estimate for any workplace exposure based on self-respondent information only was still significantly elevated (odds ratio, 1.35; 95% confidence interval, 1.01 to 1.79).

These data, together with the exposure information offered from studies such as that of Hammond and colleagues, reinforce the health concerns associated with involuntary workplace exposures to ETS that have formed the basis for recent public policy initiatives to restrict workplace smoking.

Peggy Reynolds, PhD
Julie Von Behren, MPH
California Department of Health Services
Emeryville
Elizabeth T. H. Fontham, PhD
Pelayo Correa, MD
Louisiana State University Medical Center
New Orleans
Anna Wu, PhD
University of Southern California School of Medicine
Los Angeles
Patricia A. Buffler, PhD
University of California School of Public Health
Berkeley
Raymond S. Greenberg, MD, PhD
Medical University of South Carolina
Charleston

1. Hammond SK, Sorensen G, Youngstrom R, Ockene JK. Occupational exposure to environmental tobacco smoke. *JAMA*.1995;274:956-960.
2. Fontham ETH, Correa P, Reynolds P, et al. Environmental tobacco smoke and lung cancer in nonsmoking women: a multicenter study. *JAMA*. 1994;271:1752-1759.

In Reply.—Dr Ogden exaggerates the impact of nicotine reemission. The concentration of nicotine in unoccupied settings is negligible, and neglecting it introduces only a minor error, whereas including the entire sampling time would grossly underestimate the true exposure of workers. Although the details of nicotine adsorption and reemission are not known, the rapid decay of nicotine has been widely reported.[1,2] The average concentration measured over the full week may be expressed by the following formula:

$$C_{week}=([45\ h\times C_{work}]+[5\ h\times C_{decay}]+[70\ h\times C_{night}]+[48\ h\times C_{weekend}])/168\ h$$

where h indicates hours; C_{week}, average nicotine concentration over a full week; C_{work}, average nicotine concentration during work; C_{decay}, average nicotine concentration during the first hour after work; C_{night}, average nicotine concentration overnight; and $C_{weekend}$, average nicotine concentration over the weekend.

Data collected in a realistic setting, the inside of a furnished trailer,[2] indicate that nicotine decays rapidly, to 10% of the initial level within 30 minutes and 2% in 50 minutes, and nicotine concentration does not increase subsequently (D. J. Eatough, oral communication, December 5, 1995). Using those data to calculate true workday concentration and accounting for the residual nicotine in the air, one can calculate that the values reported in our article should be reduced by less than 5%.

Although nicotine decayed less rapidly in a stainless steel chamber, it reached 10% of its initial value in 1.5 hours and 5% in 6 hours.[1] Even if the workplace is more like a stainless steel chamber (unlikely), if the overnight decay and reemission led to concentrations 10% of the workday levels, and if weekend values were half that ($C_{night}=0.1\ C_{work}$; $C_{weekend}=0.05\ C_{work}$; and $C_{decay}=0.5\ C_{work}$), the reported workday concentrations should be reduced by 20% (not the 73% suggested by Ogden). Our own tests indicate that there is no problem with adsorption and reemission of nicotine to the sampler. Ogden's finding may be an artifact related to the contamination he and Maiolo reported on all their blank filters.[3]

Personal sampling, although not feasible in this study, is usually preferable to stationary sampling, which generally underestimates occupational exposures. Since only nonsmokers' workstations were included in our analysis, the ETS sampled is attributable to far-field exposure only, while personal samples would be expected to be higher, as nonsmokers also moved into smokers' areas (near-field exposure) during the workday. Therefore, the use of area samples underestimates the true exposure of nonsmokers.

The clear association between workplace ETS exposure and lung cancer reported by Dr Reynolds and colleagues is entirely congruent with the levels of exposures we reported. Both the exposures of workers in settings where smoking is allowed and their lung cancer risks are comparable with those of people exposed only in the home.

When smoking is allowed in the workplace, nonsmokers may be exposed to hazardous levels of ETS. This was demonstrated by measurements at 25 workplaces and confirmed by the report of Reynolds et al on the association of lung cancer with workplace ETS exposure.

S. Katherine Hammond, PhD
University of California School of Public Health
Berkeley

1. Nelson PR, Heavner DL, Collie BB, Maiolo KC, Ogden MW. Effect of ventilation and sampling time on environmental tobacco smoke component ratios. *Environ Sci Technol*. 1992;26:1909-1915.
2. Tang H, Eatough DJ, Lewis EA, et al. The generation and decay of environmental tobacco smoke constituents in an indoor environment. In: *Measurement of Toxic and Related Air Pollutants: Environmental Protection Agency/Air and Waste Management Association International Symposium*. Pittsburgh, Pa: Air and Waste Management Association; 1989:596-605.
3. Ogden MW, Maiolo KC. Comparative evaluation of diffusive and active sampling systems for determining airborne nicotine and 3-ethenylpyridine. *Environ Sci Technol*. 1992;26:1226-1234.

Polyneuropathy After Mechanical Ventilation

To the Editor.—The article by Dr Leijten and colleagues[1] merits comment. The authors studied 50 patients receiving mechanical ventilation for more than 7 days and reported a high incidence of what they define as critical illness polyneuropathy. However, it is likely that a subset of the patients they reported actually had acute quadriplegic myopathy, which is an increasingly recognized disorder that produces prolonged neuromuscular weakness in the intensive care unit and may be confused with critical illness polyneuropathy.

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